Clinical Section

The Diagnosis and Treatment of Purpura

by Lennox G. Bell, M.D., M.R.C.P. (Lond.)

Spontaneous haemorrhage into the skin or from a mucous membrane is one of the most urgent signs in clinical medicine. It is a sign which calls for an immediate and exhaustive search for the underlying cause. In a few cases the causative factor may prove to be some innocuous condition; in others, the appearance of minute and scattered petechial spots may be a harbinger of certain death. In between these extremes, the proper investigation of purpura very often points the way to a curative method of therapy. For these reasons, every practitioner of medicine should have a clear conception of the mechanisms which bring about spontaneous haemorrhages and of the causes which may underlie them.

Strictly speaking, the term purpura refers merely to the appearance of haemorrhages into the skin, and includes all gradations from petechia, to large ecchymosis or even haematomata. However it has become customary to include under the general designation of purpura, a heterogeneous group of diseases characterized by the extravasation of blood into skin, subcutaneous tissues and mucous membranes. In recent years many authors have adopted the more general term "Haemorrhagic Diathesis" to designate this group of diseases.

The Mechanism of Purpura:

Generally speaking, there are three chief factors which may precipitate spontaneous bleeding into skin or mucous membranes—

1. A decreased number of blood platelets.
2. Increased permeability of capillary walls.
3. Deficiency in the elements of clot formation.

As pointed out by Perlman and Fox[1], it seems logical to construct a classification of haemorrhagic diseases based upon these three fundamental disturbances.

Let us examine each of these factors in turn.

1. Thrombocytopenia:

Clinical interest in the blood platelets dates from 1887 when Denys[2] was able to demonstrate a decrease in circulating platelets in Werlhof's type of purpura. Since that time it became customary to divide all forms of purpura into a thrombocytopoenic group and a non-thrombocytopoenic group, and gradually it became evident that those with deficient platelets included not only the idiopathic type of Werlhof, but also a symptomatic type due to a variety of causes.

Blood platelets arise in the bone marrow by a process of "budding off" from the megalocytes. Normally they number from 250,000 to 500,000 per cu. mm., and are probably destroyed by the spleen. The count is subject to a fairly wide diurnal variations and may be considerably influenced by such factors as exercise, temperature changes, and by menstruation. The platelets play an important rôle in the prevention of haemorrhage. When a vessel wall is injured platelets disintegrate producing thrombokinase, which brings about the conversion of prothrombin to thrombin in the presence of free calcium ions, and thus the coagulation process is initiated. The substance thrombokinase is probably produced also by tissue juices. The platelets tend to agglutinate and adhere to damaged capillary epithelium and arrest haemorrhage in this way. They also bring about firm retraction of formed blood clot.

Reduction of blood platelets below 40,000 per cu. mm. usually causes spontaneous haemorrhage. While this generalization does not always hold true as will be pointed out later, nevertheless the most important step in the investigation of purpura is to determine whether or not thrombocytopenia is present. If platelets are reduced much below normal figures the following will occur:

1. Prolonged bleeding time.
2. Normal coagulation time.
3. Delayed or absent clot retraction.
4. Positive Capillary Resistance Test.

In Haemophilia the platelets are normal in number but may be abnormally resistant and therefore coagulation does not take place—the platelets retain their power of adhering to a minute wound hence normal bleeding time—but they fail to disintegrate and produce thrombokinase—with prolongation of the coagulation time.

Reduction in blood platelets may be caused by conditions favoring diminished production as in aplasia of the functioning bone marrow; or in replacement of the marrow by leukaemia, metastatic malignancy, etc.; by excessive destruction of circulating platelets by the spleen; or possibly by the occurrence of multiple small haemorrhages when platelets are used up in adhering to the damaged capillary endothelium.

2. Increased Permeability of Capillaries:

Increased permeability of capillary walls is the second factor leading to purpura. This may be brought about by infections, toxic or allergic damage, or by vitamine deficiencies. In infective states, the capillaries may be damaged by bacterial emboli lodged in them as in septicaemia,

subacute bacterial endocarditis, and typhoid; or the wall may be weakened by the direct action of toxins as in meningitis. Certain drugs, notably quinine, copaiba, may exert a toxic effect upon the capillaries, and the toxins of kidney disease may produce purpura even in the absence of marked nitrogen retention. Allergy may cause increased capillary permeability—as seen in the diseases of the Erythema group, which may vary in intensity from simple erythema, urticaria, or erythema multiforme where serum alone exudes into the tissues, to the Anaphylactoid group of purpuras where urticaria, joint pains, visceral manifestations due to exudation into the walls of hollow organs, or purpura and bleeding from mucous membranes may occur in any combination. Finally, a deficiency of vitamine C or vitamine P, which apparently are necessary to maintain the integrity of the capillary wall, may give rise to bleeding as seen in scurvy.

3. Deficiency in the Elements of Clot Formation:

The haemorrhagic conditions in this group are, aside from haemophilia, all due to an increase in the prothrombin time of the blood. Thus, lack of vitamine K in the diet, deficient absorption from the intestine as seen in obstructive jaundice, biliary fistula and in some cases of idiopathic steatorrhea, diffuse diseases of the liver, and haemorrhagic disease of the newborn, are all due to prothrombin deficiency.

Analysis of 58 Cases of Purpura

(Vitamin K deficiency not included)

A—Purpura due to decrease in blood platelets:

Essential Thrombocytopenic Purpura 10

Symptomatic Thrombocytopenia:

1. Depression of Marrow Activity (Aplastic Anaemia)—2.
2. Replacement of marrow by
   (a) Leukaemia—6.
   (b) Multiple Metastasis—1.
   (c) Hodgkin's Disease—2.
3. Gaucher's Disease and Lipoid Dystrophies—0.
4. Drug intoxications due to
   (a) Gold—0..
   (b) Sedormid—0.
   (c) Arsenicals—0.
   (d) Sulphonamides—2.
5. Infections—2.
6. Allergic Purpura—2.

B—Purpura due to changes in Capillary Walls:

Anaphylactoid Purpura—
Purpura Simplex—4.
Schonlein's Purpura—3.
Henoch's Purpura—4.
Infections—14.
Nephritic Toxins—2.
Drug intoxications—2.
Vitamine deficiencies—1.

Thrombocytopenic Purpura:

In the study of any case of haemorrhagic disease it is imperative to determine whether or not the blood platelets are diminished. This is best accomplished by an actual count of the platelets although a careful examination of a stained blood smear will often give a rough idea of the number present. A prolonged bleeding time with normal coagulation time, delayed clot retraction and a positive Hess Capillary test will complete the evidence for Thrombocytopenia. Once this has been established one must make a clear-cut decision as to whether one is dealing with a Primary or a Secondary type of the disease—because very often the diagnosis of Primary thrombocytopenic purpura calls for a life-saving splenectomy, whereas such a procedure in any of the secondary varieties might prove disastrous. The history must be analyzed for the occurrence of recent infections, and for the ingestion of those drugs known to depress the platelet count. The blood must be examined carefully for evidences of aplastic anaemia, leukaemia or bone marrow disease. In this regard the criteria for the diagnosis Primary Thrombocytopenic Purpura as laid down by Wiseman, Doen and Wilson[3] are very useful.

1. There must be spontaneous purpura and/or free bleeding from mucous membranes.
2. The blood platelets must be substantially decreased in numbers, less than 100,000 per cu. mm. of blood.
3. The clotting-time and prothrombin-time must be within normal limits.
4. The anaemia and leukocyte count must not be out of proportion to the amount of bleeding.
5. There must be no pathological cells in either the blood or the bone marrow.
6. There must be no appreciable enlargement of the lymph nodes or the spleen.

In Primary Thrombocytopenic Purpura no adequate explanation for the low platelet count has yet been advanced. The most probable mechanism is that of Kaznelson who believed that the spleen destroys an excessive number of platelets in this condition. The prompt rise in blood platelets following splenectomy makes this hypothesis seem reasonable. Some observers feel that the spleen produces a hormone which depresses the formation of platelets in the bone marrow, but no histological evidence of depression of megakaryocytes has yet been demonstrated. In 1938 Troland and Lee[4] prepared an acetone extract from the spleens of their patients with Primary Thrombocytopenic purpura, which they claimed caused a marked reduction of the circulating platelets in experimental animals. Several workers have since failed to substantiate these claims.

The exact mechanism of the bleeding tendency in this disease remains obscure. Some believe that the fundamental defect is capillary

damage and that the platelets are used up in plugging up the gaps so caused. This, it is argued, would explain why in certain cases bleeding may occur when the platelet count is very slightly below normal, whereas others show no haemorrhage when the count is as low as 10,000 per cu. mm. The prolonged bleeding time and the positive Hess Capillary test are also advanced by some authors as evidences of capillary damage, but such damage may well be the result rather than the cause of the platelet deficiency.

Primary Thrombocytopenic purpura may evidence itself at any age and may pursue an acute or chronic course. It is important to note that the initial episode may be the only one to occur during a lifetime, especially in young children, although the natural history of the disease in untreated cases is to run a cyclic course marked by exacerbations and remissions. Haemorrhage may occur both in skin and from mucous membranes, but not infrequently it is manifested by recurrent, unexplained bleeding from one source. This is particularly true of cases which begin with severe menorrhagia in young women. In one of our cases, purpura with menorrhagia occurred at each menstrual period, and a drop of platelets to 40,000 was demonstrated two days before each period, rising to normal levels during the following two weeks. Not infrequently although thrombocytopenia is constantly present no purpuric manifestations or haemorrhage occur unless some injury is sustained. This occurred in two of our cases. In another, purpura only appeared during infections.

Treatment of Primary Thrombocytopenic Purpura

1. **Medical Treatment**—It is difficult to evaluate medical treatment in any disease which is marked by spontaneous remissions. A host of remedies have been suggested in the disease under discussion, of these many may be disposed of in a word. Thus cevitamic acid, the fat-soluable T-factor of Schiff, found in sesame oil, ultraviolet light, congo red, and intravenous calcium have all proved ineffective. However, it is usually advisable to tide the patient over an acute attack before performing splenectomy and for this purpose several methods have been employed. Irradiation of the spleen has been advocated by several authors but usually proved quite ineffective. Snake venom has been used by Peck and Rosenthal[5] with encouraging results, while others have found it useless. The use of a skin test to snake venom is used by Peck and Rosenthal as a guide to prognosis. They claim that patients treated with snake venom without a reversal of the skin test from positive to negative are likely to obtain little benefit from surgery. This observation needs further confirmation.

The use of Parathyroid Extract was suggested by Lowenburg and Ginsbery[6] in 1932. Two further cases of success by this form of therapy have since been published. One of our cases failed to respond to parathormone and subsequently died of cerebral haemorrhage.

Transfusion is undoubtedly the best method of controlling haemorrhage and carrying the patient until a spontaneous remission sets in, so that the surgical risk of splenectomy may be considerably lessened. As reported by Whipple and others the mortality from splenectomy in the acute stage may be as high as 87 per cent.

In suitably selected and prepared cases the results from splenectomy are excellent, but the selection of a surgeon is just as important as the selection of suitable cases. Usually bleeding stops dramatically when the pedicle of the spleen is tied off and the platelets begin to rise almost immediately. The risk of surgical haemorrhage is not so great as might be supposed, and the operative mortality, in good hands, should not exceed 5%, although the patients frequently run a stormy postoperative course. In our series 5 patients underwent operation with no deaths and prompt recovery in all.

Anaphylactoid Purpuras:

The group of Anaphylactoid Purpuras — sometimes called Haemorrhagic Capillary Toxicosis, are due to capillary damage by an allergen or infective toxin and are closely allied to urticaria and Erythema Multiforme. These forms are usually preceded by fever and general malaise. Joint pains are very common and the purpuric eruption is often combined with urticaria. In children exudation into the intestinal wall may give rise to colic and melaena and if these symptoms precede the erruption—intussusception or appendicitis may be closely simulated. Joint pain and swelling occur in the Schonlein Form and may be confused with rheumatic fever. In severe cases the capillaries of the glomerular tufts may be affected by the toxic process and glomerular nephritis results. The disease is usually self-limiting and no treatment is required except where bleeding is excessive. Reports of the successful administration of Vitamine P have recently appeared. Within the last few weeks I have used Vitamine P in a case of Schonlein's purpura with apparently good results.

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Abstract

Mesenteric Thrombosis—Recovery

by R. G. Greer, M.D.

Assistant Surgeon, Children's Hospital, Winnipeg

This case is of interest for the following reasons:

1. Complete recovery;
2. Rare condition in young woman with no evidence of Heart Lesion or Arteriosclerosis.
3. X-ray examination negative.

The patient, E.H., an unmarried woman of 37 years, was first seen in consultation with Dr. E. Steele, March 6, 1942, at 11:30 a.m. The history was as follows: the patient had awakened at 7 a.m. and on arising she was seized with a very sharp general pain in the abdomen. This was followed by nausea and she attempted to vomit but only brought up mucus secretion. From then on the pain continued severe in the paraumbilical region and did not radiate. She had her last bowel movement the morning before, March 5, 1942.

Genitourinary and Menstrual history negative. Patient had been troubled with constipation. In 1930 she had suspension of uterus and appendectomy.

Physical Examination:

On examination at 11:30, about 4 hours after onset of pain the patient looked ill. She had definite facies Hippocratica. The patient was lying on her back, face and lips were pale, pulse was 90 per minute, temperature was subnormal, 97.2. Blood pressure 115/70.

Heart Sounds: were regular and rhythmic, and no murmurs present.

Abdomen: There was a slight swelling of the abdomen in the right lower quadrant. Here there was resistance and tenderness to palpitation and an ill-defined mass could be felt.

Vaginal examination: severe pain elicited in the right fornix.

The impression at this time was a twisted ovarian cyst. The patient was given an **S.S. enema.** The enema returned ineffectual except for an odd fleck of stool and a small amount of flatus.

X-Ray: Flat plate of abdomen, negative.

Report by Dr. D. Wheeler: "Some gas is seen in transverse colon. This is not sufficient gas to indicate an obstruction. No dilated loop of small bowel is seen.

Urinalysis: Trace of albumen present; negative for sugar.

Microscopic: frequent red cells, no pus cells or casts.

Blood Examination: Hb 89% W.B.C. on admission 11,700.

At 2 p.m. patient showed no improvement and when a rectal tube was inserted she passed 150 c.c. liquid stool and some flatus.

At 4 p.m. another enema was given. Again there was a liquid return and some flatus. Patient continued nauseated. The leucocyte count, repeated, had risen to 21,200. Operation was advised.

Operation at 6 p.m.

Cyclopropane and Oxygen anesthetic was given and the abdomen opened by a lower right paramedian incision.

On opening the abdomen a large quantity of sanguinous fluid poured out and three large coils of black gangrenous small intestine, lower jejunum and upper ileum, presented. The fluid was aspirated and the affected bowel brought out of the wound. The anaesthetist was instructed to increase the amount of oxygen in his anaesthetic and an attempt was made to find the source of the disease, but there was no evidence of a twist or kink to be found. The increased oxygen had no effect on the gangrenous section so resection was necessary. About three feet of bowel with mesentry was resected.

A side-to-side anastomoses was performed in the usual manner. An enterostomy tube was then buried in the serous coat of the proximal bowel, inserted through the junction point into the healthy bowel distally. This was to insure against any contraction of my anastomosis and to allow feeding of the patient. The tube was brought out of the abdomen through a separate stab incision. About 8 grams of Sulfanilimide powder was used along the suture lines.

The paramedian wound was closed without drainage. The operation required about 1¾ hours, and the patient's condition was good.

Post-operative Course:

Upon returning to the ward the patient was given 2000 c.c. of 5% glucose and saline intravenously. Pure oxygen was given almost continuously. Nothing was given by mouth.

The following day the patient was given all nourishment (Normal Saline, 10% glucose) through the enterostomy tube and the first 24 hours received about 3000 c.c. Thereafter it was limited to 2400 c.c. in 24 hours. At the same time Sulfanilimide, 10 grains t.i.d. and tablets of Vitamin C were introduced into the bowel through the tube.

On the afternoon of March 7, 1942, the patient began to feel nauseated and vomited. Nasal suction was established with immediate relief. Fowler's position was maintained throughout.

From the seventh to the twenty-fourth day the temperature varied between 98-101 and the pulse between 90-100. This as time went on proved to be due to wound infection and it was this continued sepsis which ten days after operation reduced her Hb to 49% and made it necessary to give two blood transfusions.

Up to the fifth day when the nasal catheter was finally removed the patient received all nourishment and medication through the Enterostomy tube. Because of the marked loss of bile through the Nasal Suction Tube, some of this was filtered and introduced into the enterostomy.

After this period, she gradually began to take nourishment by mouth, normal bowel movements were established, wound infection had cleared up and she left the hospital in perfect health on April 8, 1942.

Follow-up history and X-rays showed normal bowel function.

Editorials and Association Notes

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Annual Meeting

September 23rd, 24th and 25th, 1942

At a recent meeting of the Executive of the Manitoba Medical Association, there was considerable discussion regarding the programme and entertainment for the forthcoming meeting. It was felt by the Executive that the entertainment should be curtailed this year. It was accordingly decided to omit the usual dinner-dance. A public meeting will be held in the main Auditorium on the evening of September 24th, 1942.

Tentative arrangements for this public meeting are under way. There will be four brief addresses on health matters of interest to the general public. The speakers will likely be:

Dr. A. E. Archer, President of the Canadian Medical Association.

Dr. F. Gillespie of Edmonton, Alberta.

Dr. J. W. Scott of Edmonton, Alberta.

Dr. F. W. Jackson, Deputy Minister of Health for the Province of Manitoba.

It is hoped that the public meeting will receive a good response.

North West Medical Society

That most active district medical society, the North Western, held a meeting at the Sacred Heart Hospital, Russell, on August 19th, which attracted medical men and their wives from Neepawa, Virden, Hamiota, Birtle, Shoal Lake, Miniota, Langenburg, Roblin, Russell, Minnedosa, Rossburn, Angusville, Spyhill and Binscarth. Dr. John Skafel of Minnedosa presided, and Dr. E. D. Hudson, a senior member of the Canadian Medical Association, was secretary. The guest speakers, all from Winnipeg, were: Dr. Neil John MacLean, who spoke on Hernias; Dr. W. A. Gardiner, who spoke on Club Feet, and Dr. J. C. Hossack on Nervous Diseases.

Wives of the out-of-town doctors were guests at the home of Mrs. T. I. Brownlee. The visiting doctors, numbering 28, were entertained at dinner in the Queen's Hotel. September meeting in Winnipeg.

Free Postage on Library Loans

The MEDICAL LIBRARY has been granted a reduced postal rate to be used for all loans of BOOKS or PERIODICALS mailed to places within the province of Manitoba.

From now on all the borrower has to do is to affix THE LABEL supplied by us and follow the instructions printed thereon. NO postage then need be paid.

Abstract

Sulfathiazol Powder for Bedsores:

Goodman and Corsaro (Ohio State M.J. 1941, 37, 956) report that five stubborn cases healed rapidly after daily application of Sulfathiazole powder to the ulcer with a salt shaker. Carbon light was also used. This method has also been used with success on other types of ulcers, and sinuses.

Halitosis:

Crohn and Drosd (J. Am. M. Ass. 1942, 117, 2242) state that halitosis not due to local causes responds to a low fat diet (50 gms. daily). The Chinese think all eaters of milk and butter smell like cows. Experiments were done on garlic. It was concluded that garlic is absorbed in the intestine and excreted in the liver bile for several days. During this time the odor of garlic is conveyed by the blood to the lungs, and thence by the expired air to innocent bystanders.

For immediate, if somewhat temporary relief from garlic or onion breath, the method advocated in J. Am. M. Ass. 1935, 104, 2162 may be followed. Half a five-grain Chloramine (Chlorazine) tablet dissolved in an ounce of water when used as a mouth wash destroys any essential oils in the mouth at the time. The abstractor can state from personal experience that a green onion flavor can be driven from the mouth if this mouth wash is used two-hourly.

Commercial Exhibitors at Annual Meeting

Doctors will have the opportunity of visiting the following business organizations who will have displays at our Annual Meeting. Officials and representatives will be in attendance to give information on the products displayed. The following list includes only those firms who had made reservations at the time of going to press:

Brathwaites Limited	Ingram and Bell
Carnation Company	Eli Lilly
Ciba Company	Winthrop Chem. Co.
Fisher & Burpe Ltd.	John Wyeth & Brother
H. G. Fischer Inc.	Winnipeg Drug Co.

Also we wish to acknowledge the reservation of "Display Cards from the following firms:— Chas. E. Frosst Co.; Robert Ramsay, The Stevens Companies, Victor X-Ray Corp.

Obituaries

Dr. John Sutherland Matheson

Dr. John Sutherland Matheson, a pioneer surgeon of Brandon, died at his home on July 27 after a long illness.

Born at Woodstock, Ont., in 1874, he came with his parents to the new settlement in Brandon in 1883. At the age of 20 he graduated in medicine from Toronto University and after post-graduate work in Edinburgh and Chicago, he began practice in Brandon in 1895. Here he threw himself into the life of the community. For 24 years he was a member of the school board, serving several terms as chairman. He was a member of the honorary attending staff of Brandon General Hospital and a member of the Board of Directors. During the first World War he served with distinction, returned with the rank of Lieutenant-Colonel, and became chief medical officer at M.D. 10. He was a fellow of the American Colege of Surgeons and of the Royal College of Surgeons of Canada.

His wife, a son—Dr. Murray S. Matheson, and a daughter, Mrs. George Davidson of Vancouver, survive him. His death will be mourned by a wide circle of friends throughout Western Canada.

Dr. J. B. Cloutier

Dr. J. B. Cloutier of Letellier, died at his home on August 5 of cerebral haemorrhage. Born at Riviere du Loup, Quebec, fifty-two years ago, he graduated in medicine in 1916 and came to Manitoba to practice in Letellier. He is survived by his widow and five children, of whom the oldest son is a member of the R.C.A.F., in Edmonton.

First Annual Meeting

Manitoba Health Officers' Association

Tuesday, September 22nd, 1942

a.m.—

9:15 Registration and film on Health Subjects.

9:30 E. S. Bolton, M.D., D.P.H., Director of Health, City of Brandon, Man.—"Experiences in the Control of Diphtheria and Scarlet Fever by Immunization."

Discussion opened by O. J. Day, M.D., director of City of Winnipeg Health of Children Services.

10:00 Mr. J. Foggie, Chief Provincial Sanitary Inspector—"Problems in Sanitation, particularly re Septic Tanks and Cesspools."

10:30 Questions and Answers.

11:00 F. Walkin, M.D., Ashern, Man.—"Problems of the Part-time Health Officer in Northern Manitoba."

The morning session will be held in the Auditorium of the Department of Health Building, 320 Sherbrook Street, Corner Portage Avenue

12:30 Luncheon—(Place to be announced). The Hon. J. O. McLenaghan, Minister of Health and Public Welfare, will give an address.

Those attending will be guests of the Department of Health.

p.m.—

2:00 Election of Officers and Business.

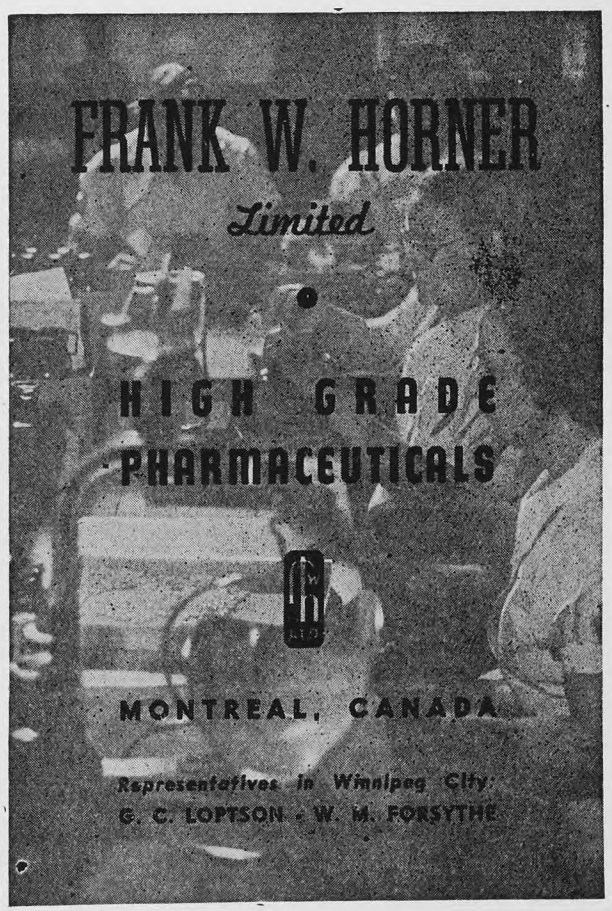
2:30 President's Address.

2:45 Discussion re Remuneration of Part-time Health Officers.

3:15 F. Purdie, M.D., Griswold, Man.—"Random Experiences of a Part-time Health Officer."

3:45 C. E. Mather, M.D., D.P.H., of the Department of Health — "How the Part-time Health Officer can Readily Improve His Work."

4:15 C. R. Donovan, M.D., D.P.H., Director of Disease Prevention— "Information Regarding the Present Status of Disease Prevention."



Personal Notes and Social News

Dr. and Mrs. W. T. Dingle arrived from Scotland to be the guests of Mr. and Mrs. J. R. Dingle, Winnipeg.

◈ ◈

Dr. and Mrs. Harry Benwell and their daughter, of Grand Forks, N.D., were recent visitors to Winnipeg.

◈ ◈

Dr. C. H. Moore, recently a resident surgeon at the St. Boniface Hospital, has joined up for active service in the navy.

◈ ◈

Dr. C. V. McClelland was married July 18th to Annie L. Lang. Dr. and Mrs. McClelland will reside in Pine Falls, Man.

◈ ◈

Dr. and Mrs. R. L. Howden of Norwood, Man., are celebrating the birth, on July 29th, of a son (Robert Campbell), at St. Boniface Hospital.

◈ ◈

Capt. J. F. A. McManus, R.C.A.M.C. (overseas) and Mrs. McManus are celebrating the birth of a son (Anthony Edward) on July 30th, at Grace Hospital.

◈ ◈

Dr. and Mrs. Eyjolfur Johnson of Selkirk, Man., are receiving congratulations on the birth of a daughter on July 27th, at the Winnipeg General Hospital.

◈ ◈

Dr. Roger Knipe of Vancouver, son of the late Dr. G. W. Knipe, and his mother, were recent visitors to Winnipeg, where they spent a few days renewing old acquaintances.

◈ ◈

Flight-Lieut. R. G. Cadham, R.C.A.F. (overseas), and Mrs. Cadham announce the arrival of a son to be named Fred James, at the Winnipeg General Hospital on July 24th.

◈ ◈

Dr. Donald J. Hastings, elder son of Dr. and Mrs. H. E. Hastings, is to be married in Holy Trinity Church, September 12th, to Isobel Mary, only daughter of Mr. and Mrs. Charles Carver of Winnipeg.

Major William J. Boyd, who went overseas as second in command of a light field ambulance unit, has been promoted to the rank of Lieut.-Colonel and placed in charge of No. 4 Field Ambulance Brigade.

◈ ◈

Dr. Isabelle McTavish, medical missionary to China for 25 years, recently returned safely to Canada. Dr. McTavish graduated from the Manitoba Medical College in 1915, and her home is in Newdale, Man.

◈ ◈

Lieut. Sam Rusen, R.C.A.M.C., was married on August 11th to Edith, eldest daughter of Dr. and Mrs. S. Kobrinsky of Winnipeg. After a short honeymoon Dr. and Mrs. Rusen will reside temporarily in Brandon.

◈ ◈

Dr. Digby Wheeler was awarded the Shaughnessy Cup for the most beautiful home grounds in all classes, in the Greater Winnipeg garden competition, sponsored by the Manitoba Horticultural Society.

◈ ◈

Capt. John D. Leishman, son of Dr. and Mrs. A. G. V. Leishman of Winnipeg, was married June 20th, in St. Mary's parish church, Harsham County, Essex, England, to Nursing Sister Gertrude Eileen Lake of St. Johns, Newfoundland.

◈ ◈

Dr. Irwin H. Mazerovsky has been appointed provincial coroner at Churchill.

◈ ◈

Dr. J. Roy Martin of Neepawa and Dr. A. F. Menzies of Morden have been appointed by the Manitoba Government to the provincial Board of Health. They will fill the vacancies created by the deaths of Dr. G. W. Rogers, Dauphin, and Dr. J. S. Matheson, Brandon.

◈ ◈

Three Medical Musketeers—Drs. B. E. Loadman, J. E. Hudson and C. E. Corbett, three college chums who graduated in the same class from the Manitoba Medical College, joined the same army unit, went to Camp Borden and returned to Camp Shilo as lieutenants, have all been promoted captains and attached to the 3rd Field Ambulance, R.C.A.M.C.

Department of Health and Public Welfare

COMMUNICABLE DISEASE REPORT

June 18th to July 15th, 1942

MEASLES: Total 239—Winnipeg 92, St. Andrews 19, St. Boniface 17, St. Vital 12, Minitonas 8, Kildonan East 6, Piney 7, Selkirk Town 5, Tuxodo Town 4, Deloraine Town 3, Portage la Prairie City 3, Winchester 3, Brandon 2, Ellice 2, St. James 2, Tache 2, Arthur 1, Beausejour Town 1, Fort Garry 1, Kildonan West 1, Norfolk North 1, Stonewall Town 1, Ste. Anne 1, Wallace 1. Late Reported: Piney 28, Deloraine Town 7, Fort Garry 2, St. Vital 2, Winchester 2, Brandon 1, Lorne 1, Tache 1.

CHICKENPOX: Total 124—Winnipeg 61, Napinka Village 12, Virden Town 10, Neepawa Town 8, St. Boniface 7, Arthur 4, Rosser 3, Transcona 2, Brenda 1, Elkhorn Village 1, Glenwood 1, Hillsburg 1, Kildonan East 1, Kildonan West 1, Melita Town 1, Minnedosa Town 1, Souris Town 1, Stonewall Town 1, St. James 1, St. Vital 1, Tuxedo Town 1, Woodworth 1, Unorganized 1. Late Reported: Brooklands Village 1, Springfield 1.

MUMPS: Total 114—Portage la Prairie City 28, Brandon 24, Winnipeg 18, St. Vital 7, Portage la Prairie Rural 5, St. Boniface 5, Grandview Rural 3, Tuxedo Town 3, Unorganized 2, Daly 1, Dauphin Town 1, Flin Flon 1, Hamiota Rural 1, Norfolk North 1, Transcona 1, Wallace 1, Whitehead 1. Late Reported: St. Andrews 3, Transcona 3, Portage la Prairie City 1, Brandon 1, St. Vital 1, Virden Town 1, Unorganized 1.

TUBERCULOSIS: Total 83—Winnipeg 35, St. Vital 4, St. James 3, Dauphin Town 2, De Salaberry 2, Kildonan North 2, Siglunes 2, St. Boniface 2, St. Clements 2, Unorganized 2, Argyle 1, Brandon 1, Coldwell 1, Cornwallis 1, Dauphin Rural 1, Fort Garry 1, Kildonan East 1, Kildonan Old 1, Kildonan West 1, La Broquerie 1, Mossey River 1, Rhineland 1, Roland 1, Rosedale 1, Selkirk Town 1, Shellmouth 1, Shell River 1, Springfield 1, Swan River Rural 1, Ste. Anne 1, St. Laurent 1, St. Paul East 1, Ste. Rose Rural 1, Thompson 1, Transcona 1, Turtle Mountain 1, Woodlands 1.

SCARLET FEVER: Total 79—Winnipeg 35, Brandon 5, Portage la Prairie Rural 5, Cornwallis 2, Fort Garry 2, Norfolk North 2, Portage la Prairie City 2, Birtle Rural 1, Dauphin Town 1, Elton 1, Hillsburg 1, Kildonan East 1, Kildonan North 1, Kildonan West 1, Sifton 1, Springfield 1, Swan River Rural 1, Tache 1, Transcona 1, Tuxedo Town 1. Late Reported: Dauphin Rural 10, Fort Garry 1, Harrison 1, Springfield 1.

WHOOPING COUGH: Total 16—Souris Town 3, Kildonan East 2, Virden Town 2, Winnipeg 2, Brandon 1. Late Reported: Gilbert Plains Rural 4, Brandon 1, Gilbert Plains Village 1.

DIPHTHERIA: Total 15—Tuxedo Town 3, Winnipeg 3, St. Vital 2, Cornwallis 1, Hanover 1, MacDonald 1, Neepawa Town 1, Richot 1, St. Boniface 1, Tache 1.

ERYSIPELAS: Total 10—Winnipeg 6, Hanover 1, Rockwood 1, St. Andrews 1, Transcona 1.

GERMAN MEASLES: Total 9—Brandon 4, Kildonan West 3, Norfolk North 1, Portage la Prairie Rural 1.

ANTERIOR POLIOMYELITIS: Total 7—Gladstone Town 1, Portage la Prairie Rural 1, Unorganized 1. Late Reported: Kildonan East 1, Kildonan West 1, Fort Garry 1, Roland 1.

LOBAR PNEUMONIA: Total 3—Hanover 2, South Norfolk 1.

ENCEPHALITIS: Total 1—Westbourne 1.

SEPTIC SORE THROAT: Total 1—Whitewater 1.

DIPHTHERIA CARRIERS: Total 1—Winnipeg 1.

VENEREAL DISEASE: Total 126—Gonorrhoea 95, Syphilis 31.

TREATY INDIANS: Total 17—Tuberculosis 14, Mumps 3.

DEATHS FROM COMMUNICABLE DISEASES

June, 1942

URBAN—Cancer 50, Tuberculosis 11, Syphilis 7, Lethargic Encephalitis 3, Pneumonia Lobar 2, Pneumonia (other forms) 3, Influenza 1, Hodgkin's Disease 1, Brucellosis 1. Other deaths under one year 17. Other deaths over one year 155. Stillbirths 15. Total 266.

RURAL—Cancer 30, Tuberculosis 11, Pneumonia Lobar 5, Pneumonia (other forms) 10, Syphilis 1, Septicemia (Non-Puer.) 1, Cerebrospinal Meningitis 1, Mumps 1. Other deaths under one year 20. Other deaths over one year 141. Stillbirths 6. Total 227.

INDIANS—Tuberculosis 6 ,Influenza 2, Pneumonia (other forms) 1. Other deaths under one year 2. Other deaths over one year 5. Stillbirths 2. Total 18.

DISEASES	Manitoba June 18-July 15	Ontario June 14-July 11	Saskatchewan June 14-July 11	Minnesota June 14-July 11	North Dakota June 14-July 11
Amoebic Dysentery				8	
Anterior Poliomyelitis	3	1		3	1
Meningococcal Meningitis		12	2	1	
Chickenpox	122	878	141	148	
Diphtheria	15	8	13	8	4
Erysipelas	10	4	3	3	
Influenza		11	4	1	6
Encephalitis	1		4	2	4
Measles	195	966	48	763	44
German Measles	9	122	18		
Mumps	103	1063	341		
Scarlet Fever	66	483	85	88	21
Septic Sore Throat	1	8			
Smallpox			1	1	
Tuberculosis	83	181	60	43	16
Typhoid Fever		1		7	
Typh. Para-Typhoid		7	1		1
Undulant Fever		7			
Whooping Cough	10	227	3	139	21
Diphtheria Carriers	1				
Gonorrhoea	107	521			30
Syphilis	54	406			34

Note the eight cases of AMOEBIC DYSENTERY in Minnesota.

POLIOMYELITIS—a few cases only. To date of writing (Aug. 17) the total for Manitoba in 1942 is 28. At this date in 1941 the total was approximately 650 and the peak of the epidemic was past, but it must be remembered that the 1941 epidemic was one month earlier than is usual in Manitoba.

DIPHTHERIA is still too prevalent—15 cases in Manitoba and 13 cases in Saskatchewan for our population is definitely too high.

MEASLES, MUMPS and SCARLET FEVER all show a decline.

SMALLPOX in Saskatchewan and Minnesota. On account of the increase in travel by personnel of the army, navy and air force, not to mention of civilians, it is quite probable that *infection of this disease may be brought into Manitoba at any time.* We recommend to all Medical Health Officers and Physicians that they lose no time before vaccinating and re-vaccinating all those in their care. Don't say you were not warned!

ENCEPHALITIS—25 cases reported to date in 1942; of these eight have died, giving a case fatality rate of 31%. This is double the case fatality rate for this disease in 1941. Are we missing some cases or are they not seeing a doctor? At this time last year the epidemic was at its height and 270 cases had occurred.